

Amendments to the Specification:

Please amend the specification as shown:

- 1) Please delete the title of the invention found on page 1, lines 1-2 and replace it with the following title:

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COMPOSITIONS FOR INHIBITING THE AGGREGATION
PATHWAY OF α -SYNUCLEIN

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- 2) Please delete the text under the "Brief Description Of The Drawings" found on page 5, lines 17 thru 26 and page 6, lines 1 thru 18, and replace it with the following:

Figure 1 consists of various diagrammatic representations illustrating α -synuclein increases iron dependent toxicity wherein Fig. 1A illustrates BE-M17 cells over-expressing wildtype, A53T or A30P α -synuclein treated with varying doses of FeCl₂ for 48 hrs and the viability was determined using the MMT assay; Fig. 1B illustrates BE-M17 cells over-expressing wildtype, A53T or A30P α -synuclein treated with varying doses of FeCl₂ for 48 hrs and the viability was determined using the LDH assay; Fig 1C illustrates BE-M17 cells over-expressing wildtype, A53T or A30P α -synuclein treated with varying doses of H₂O₂ for 48 hrs and the viability was determined using the MMT assay; Fig D illustrates in a, b, c and d the sequestration of iron due to α -synuclein.; *p<0.01, + , p<0.01 by ANOVA analysis.

Figure 2 consists of diagrammatic representations illustrating that iron binds to α -synuclein wherein Fig 2A FeCl₂ quenches the fluorescence emission spectrum by tyrosine in α -synuclein (λ_{ex} =280nm); high doses of iron (>10mM) will quench tyrosine fluorescence, however proteins that bind iron exhibit quenching at much lower doses (presumably because the iron is kept near the tyrosine by the protein binding, tyrosine having a fluorescence emission spectrum that has a peak emission of 310 nm when excited at 280 nm, respectively; and wherein Fig 2B shows dose response curves for iron binding to wildtype and ΔC_{1-113} α -synuclein based on fluorescence emissions, wherein a

deletion construct lacking the last 27 amino acids of α -synuclein and analyzed binding of this construct, and a C-terminal construct of α -synuclein ΔC_{1-113} showed over a 4-fold reduction in iron binding, with an $IC_{50} = 726\mu M$ ($P<0.001$).

Figure 3 consisting of representative gels illustrating that magnesium protects against synuclein aggregation wherein Fig. 3A illustrates that magnesium converts α -synuclein to a conformation that resists aggregation and Fig 3B illustrating magnesium inhibits α -synuclein aggregation; magnesium (0.1mM) inhibits iron-induced aggregation of recombinant wild type α -synuclein in primary neurons where higher doses are needed because the cell membrane poses a barrier to passage of the ions and similar results are seen in BE-M17 cells over-expressing α -synuclein.

On page 26, line 18, after the word treatment, please insert the following: (see Fig. 1D).